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EVALUATION OF BIOCHEMICAL PROFILE IN NON TRAUMATIC COMA PATIENTS: A PROSPECTIVE HOSPITAL BASED STUDY

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Abstract:

Background: Non traumatic coma is a serious condition which is a very common presentation in emergency department. This study aimed at studying the biochemical changes in non traumatic coma patients.

Materials and Methods: This study was done in Department of General Medicine, Tertiary care hospital. The study duration is one year. A total of 50 non traumatic coma patients were included in the study based on the inclusion and exclusion criteria. All the patients demographic, clinical and biochemical data was collected. The data was expressed in number, percentage, mean and standard deviation. SPSS (16.0) version software used for statistical analysis.

Results: Maximum number of patients with age between 41-60 years. Males were more compared to females. More number of patients with metabolic disorders compared to other etiology. Increased liver enzymes, lipid profile were observed. There is a significant increase and decrease glucose, sodium levels with increased ketonebodies and uric acid levels were observed.

Conclusion: Ceribrovascular and metabolic are the most common etiology compared to others. Significant change in the biochemical parameters was observed which can indicate the severity of the condition.

Keywords: coma, hyperglycemia, lipid profile, metabolic, liver enzyme, ketone bodies

Introduction

Since the days of the Greeks, men knew that normal conscious behavior reckons on intact brain function and hence the disorders of consciousness are implied as cerebral insufficiency. Reduced and/or absences of consciousness implies the presence of severe brain dysfunction and so demands immediate attention from the physician for expectation of potential recovery. Stupor and coma mean advanced brain failure[1,2]. If such brain failure lasts for a long time the margin between recovery and development of permanent neurologic injury becomes narrower. In hospital neurology, the clinical analysis of comatose patients becomes a responsibility[3,4]. There is always urgency in a need to determine the underlying disease process causing coma and the direction in which it is evolving and also to protect the brain against irreversible or more serious damage. The terms stupor, confusion, unconsciousness, and coma have been endowed with many distinct meanings that it is almost not possible to avoid uncertainty in their usage[5]. They are strictly not medical terms but literary, philosophic and psychological ones as well. Physicians, being practical and objective for the most part, give greater plausibility to patient's behavior and response to overt stimuli than to what the patient says. So they usually give the term consciousness its commonest and simplest meaning the state of patient's awareness of self and environment and his responsiveness to external stimulus and inner need. Unconsciousness has the just opposite meaning a state of unawareness of self and environment or a suspension of those mental activities by which people are made aware of themselves and their environment, coupled with a diminished responsiveness to environmental stimuli[6]. The morbidity and mortality in non traumatic brain coma is mainly based on the etiology. It was studied that various factors can increase the duration and mortality in these patients. In these one of the common causes for non traumatic coma is metabolic changes. Changes in the glucose metabolism, liver function, ions and lipid profile can cause the prolongation of coma and mortality. The present study aimed to evaluate the biochemical changes in patients with non traumatic coma.

Materials and Methods

Study period and settings

This study was conducted in the Department of General Medicine, Tertiary care hospital. It is prospective study was done for one year. The study protocol was approved by the Institutional Research Committee (IRC) and Institutional

Human Ethics Committee (IHEC) and conducted as per protocol guidelines.

Inclusion criteria

- More than 18 years
- Coma state more than 6 hours
- No genetic brain problems
- Willing to give consent by relatives/guardian of patients

Exclusion criteria

- Preexisting traumatic coma
- Coma state less than 6 hours
- Recent brain surgery
- On anti-cancer medication for any of brain tumors
- Not willing to give consent by relatives/guardian of patients

Procedure

Total of 50 patients was included randomly based on the inclusion and exclusion criteria. All the patient's relatives/guardian was explained study protocol in understandable language and inform consent was obtained. Patients demographic (Age, gender), etiology and biochemical parameters was recorded. Liver enzymes, lipid profile, sodium, glucose, ketone bodies and uric acid was estimated by using fully auto mated analyzer. The data was collected and analyzed.

Statistical analysis

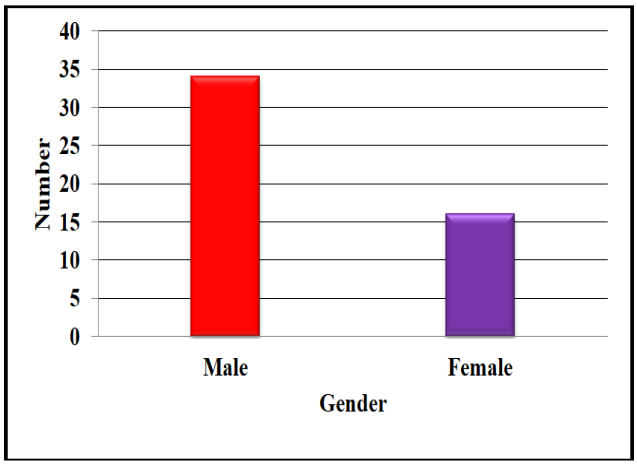
The data was expressed in number, percentage, mean and standard deviation. Statistical Package for Social Sciences (SPSS 16.0) version used for analysis.

Results

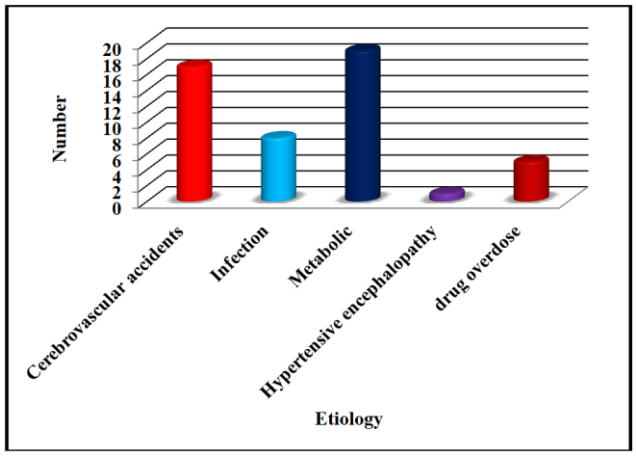
The study included patients with age less than 20 to above 80 years. 22 patients have age between 41-60 years and 61-80 years 15 peoples, 3 less than 20 and 2 more than 80 years. Maximum number patients had age between 41-60 years and minimum us above 2 (Table-1). Males (n=33) are more compared to female (n=17) patients (Graph-1). In the etiology maximum patients had metabolic compared to others and minimum is hypertensive encephalopathy. Patients with cerebrovascular accidents, infection and drug over dose was lesser compared to metabolic (Graph-2). Liver enzymes was increased compared to normal values and also serum sodium also increased (Table-2). Total cholesterol, triglycerides, LDL, VLDL was increased and HDL decreased compared to normal values (Table-3). Fasting and postprandial glucose levels were increased. Slight increase in ketone bodies and uric acid also observed (Table-4). In over all there is a increase in all biochemical parameters compared to normal lab values in non traumatic coma patients. 6 patients showed decreased in sodium levels and 5 patients showed hypoglycemia.

Table 1: Distribution of patients based on the age

Age (Years)	Number (n=50)	Percentage (%)
Less than 20	3	6.00
21-40	8	16.00
41-60	22	44.00
61-80	15	30.00
Above 80	2	4.00



Graph 1: Distribution of patients based on the gender



Graph 2: Distribution of patients based on the etiology of coma

Table 2: Mean hepatic enzyme and sodium levels in non traumatic coma patients

Observations	(MEAN±SD)
Alkaline phosphate (IU/L)	105.78±1.84
Asperate transaminase (IU/L)	29.45±2.93
Alanine transaminase (IU/L)	27.12±1.65
Total bilirubin	0.84±1.84
Conjugated bilirubin	0.23±2.94
Increased Sodium (mEq/L)	156.78±6.56
Decreased Sodium (n=6) (mEq/L)	45.89±7.45

Table 3: Mean Lipid profile levels in non traumatic coma patients

Lipid profile	(MEAN±SD)
Total cholesterol (mg/dl)	145.83±2.91
Triglycerides (mg/dl)	98.45±1.89
HDL (mg/dl)	29.45±1.34
LDL (mg/dl)	87.56±2.19
VLDL (mg/dl)	19.69±1.21

Table 4: Mean fasting, postprandial glucose, ketone bodies and uric acid levels in non traumatic coma patients

Observations	(MEAN±SD)
Fasting glucose (mg/dL)	125.89±2.78
Postprandial glucose (mg/dL)	178.76±1.34
Fasting glucose (mg/dL) (n=5)	35.78±2.98
Postprandial glucose (mg/dL) (n=5)	67.45±1.56
Ketone bodies (mmolL)	1.34±3.78
Uric acid (mg/dl)	6.2±1.45

Discussion

The present study conducted systematically to evaluate the biochemical changes in non traumatic coma patients. A total of 50 patients were included in the study. Males were more compared to females. The present study etiology showed that maximum patients had metabolic and cerebrovascular accidents for coma. Xin Y et.al., study also showed similar results. They observed that more than 40-50% had metabolic and 30-40% cerebrovascular accidents. In our study also showed similar percentage of etiology. There is a relationship between the neurological problems with liver[7]. Jones EA et.al., studied the liver enzyme levels in the coma patients. It was observed that patients with non traumatic coma showed increase in the liver enzymes compared normal peoples. It was explained that in coma there may be increased production of free radicals and ammonia. To neutralize this liver enzymes are required. It may be the reason to increase the levels of liver enzymes in non traumatic coma patients[8]. Abidullah K et.al study also showed that increased liver enzymes patients with hepatic encephalopathy. It was observed that production of more ammonia may be the one of the cause to prolong the coma. In the present study showed similar increase in liver enzymes in non traumatic coma patients. Sodium is the major extracellular cation in the body. Intra and extra cellular sodium levels are maintained by Na-K ATPase pump and total body sodium is balanced by renal excretion and secretion[9]. Martin T et.al., study showed the changes in the renal function, which can alter the excretion of sodium in urine. In the study it was observed that coma patients showed the increased sodium levels. In our study also showed similar results but few patients in the study showed decreased sodium levels. It can be understand that decreased or increased levels of sodium can prolong the coma stage. Body cells in coma patients will reduce the sensitivity to take the glucose due to reduce the insulin sensitivity. The excess glucose will leads to hyperglycemia. Increased glucose is enters into the liver and undergo lipogenesis. Due to reduction in glucose supply will stimulate the peripheral lipid catabolism. Increase the lipogenesis and lipolysis leads increase lipid profile[10]. Bewsher PD et.al., study confirmed that increased lipid profile and associated with increased glucose levels. In the present study also showed hyperglycemia with hyperlipidemia. Increased glucose undergoes ketone body formation which increases the serum ketone bodies levels. In some patients it was obsrved that decreased glucose levels which reduce the brain cell activity and can cause the coma. Uric acid is a byproduct of nucleic acids metabolism. Increased uric acid levels could be used as an independent predictor of disease progression. Uric acid levels are depends on the cell destruction in the body. They both are directly proportional each other[11]. Weilai C et.al., and Oreshnikov E et.al., studies showed minimal increase in uric acid levels and similar results was observed in the present[12,13]. The study showed that increased liver enzymes, sodium, lipid profile, glucose, ketone bodies and uric acid can be the important factors to prolong the coma stage and increase the mortality.

Conclusion

The study results conclude there is a significant change in biochemical parameters in non traumatic coma patients. These may be one of the reasons to prolong the coma state and mortality. Early correction of biochemical parameters may decrease the coma period and mortality.

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